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Editorial Comment

Changing Concepts of Cardiac Tamponade*

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In this issue of the Journal (1) appears another important contribution from the laboratory of Noble Fowler concerning the pathophysiology of cardiac tamponade. The authors attempt to identify the relative contribution of the compression of individual cardiac chambers to the overall picture of cardiac tamponade. It is now well recognized that in a variety of clinical circumstances tamponade may differ importantly from experimental cardiac tamponade in dogs and from clinical cardiac tamponade occurring after acute thoracic injury; especially in the presence of preexisting heart disease, cardiac tamponade is apt to present in an atypical way after cardiac surgery.

We have come to recognize that pulsus paradoxus may be absent in cardiac tamponade when there is preexisting elevation of the diastolic pressure of either ventricle. The syndrome of cardiac tamponade without pulsus paradoxus is best described in nephrogenic cardiac disease; in this condition tamponade may ensue in patients who already have a hypertrophied left ventricle in which diastolic pressure is greatly elevated. Similarly, severe right ventricular hypertrophy may also make resistance to filling of both sides of the heart unequal and thus abolish the conditions necessary for pulsus paradoxus to be manifest in cardiac tamponade. Pulsus paradoxus may also be absent when cardiac tamponade complicates aortic regurgitation or atrial septal defect. Low pressure cardiac tamponade is now a recognized entity, and clinicians are increasingly aware that cardiac tamponade is apt to be atypical when it complicates cardiac surgery because effusion may be localized by adhesions and the offending fluids may be partly clotted. Fowler et al. (1) approach the question of regional transmural deformation from the opposite direction, that is by measuring hemodynamic changes resulting from experimental regional cardiac tamponade. Their studies are important to the understanding both of cardiac tamponade in general and of its atypical forms, particularly localized tamponade such as occurs after cardiac surgery.

Compression of the cardiac chambers in tamponade. When the hemodynamics of cardiac tamponade were first clarified, the depression of arterial blood pressure and cardiac output, elevation of systemic and pulmonary venous pressure and appearance of pulsus paradoxus were explained as the result of a compressive force distributed equally over all the intrapericardial structures. It was subsequently appreciated that individual intrapericardial structures should not have identical ability to resist external compression and a number of later experimental results seemed to indicate that reduction of left ventricular preload, and thus stroke volume, was due to a reduction of right ventricular stroke volume occurring after compression of the thin-walled right atrium and right ventricle. For example, increasing intrapericardial pressure in closed chest dogs whose pulmonary circulation was maintained by right heart bypass did not cause arterial hypotension. Subsequently, it was shown that the right ventricular outflow tract is compressed in patients with cardiac tamponade, but not in patients with large pericardial effusion without tamponade. Later, it was demonstrated in a canine model that, when cardiac tamponade is created abruptly, left ventricular stroke volume is maintained for several beats, whereas right ventricular stroke volume declines with the first beat. Right atrial compression and right ventricular diastolic collapse are now known as highly specific and sensitive signs of cardiac tamponade, whereas compression of the left-sided chambers is much less common in tamponade. As a result of this evidence, it came to be thought that the thick-walled left ventricle is not significantly compressed by fluid exerting the increase in pressure associated with clinical cardiac tamponade.

Regional tamponade. In a series of experiments in which sutures were used to divide the pericardial space into separate compartments, Fowler et al. have studied the problem of how tamponade limited to specific regions of the heart may be relevant to the pathophysiology of cardiac tamponade. Whereas these studies confirm that arterial hypotension and reduction in cardiac output stem principally from compression of the right heart chambers, they nevertheless demonstrate that compression of the left atrium and even of the left ventricle increases the severity of cardiac tamponade.

Clinical implications. Cardiac tamponade is most often missed, and with important clinical consequences, when one or more components of the typical constellation of findings of cardiac tamponade are absent. Nowhere is this more common than in the postoperative patient. Atypical cardiac

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tamponade must be considered in every unexplained instance of hemodynamic deterioration occurring during the first 2 weeks after a cardiac operation. In this setting, pulsus paradoxus may be absent and the echocardiogram may fail to show effusion all around the heart. As this most recent study from Fowler's laboratory indicates, regional tamponade is more apt to produce serious hemodynamic consequences when the right atrium and right ventricle are compressed, but even left-sided compression may cause a significant reduction in stroke volume.

Reference

1. Fowler NO, Gabel M, Buncher CR. Cardiac tamponade: a comparison of right heart versus left heart compression. J Am Coll Cardiol 1988;12:187–93.